product that day; is that your testimony?
MR. RHEINGOLD: Objection to form.
THE WITNESS: As expressed -- I can't agree with it as you stated, but,

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Shields

basically, it's what I said. If he had not taken it that day, the causal relationship of the use of the product to the blowing of the aneurism is not changed, in my opinion.
MR. OETHEIMER:
Q. Is there anything, if I asked you to assume the correctness of his testimony that he did not take it that day, for purposes of this question, is there anything in your report, anything at all in your December 4th, 2006 report, that you would change?
A. No, except I would include that fact.
Q. That he did not take it that day?
A. That there's some confusion as to whether he took it that day or not.
Q. But if I asked you to assume, not confusion, but that for purposes of this hypothetical question, assume that it is an established fact that he did not take it that way.
Is there anything in your report that you would change?
A. Well, that's a trick question. If

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Shields

that were the fact, that would be a fact that I would have to put into the report, but would it change my conclusion, no. So to --
Q. I'm entitled to ask hypothetical questions, so I am asking you to take that it is a fact.
A. I totally understand. I'm just making myself totally clear.
It would not change my conclusion. It certainly would change the facts that I would write down.
Q. But would it affect, whether it changed your bottom line conclusion, would it affect your analysis at all?
A. No.
Q. Why not?
A. Because it would be clear to the unsophisticated, that if there were a closer temporal relation, it would be easier to understand one way or the other.
Q. What if he didn't take it that day before the stroke?
A. That's a completely different situation, and I would have to know how he was

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Shields

the day before and what kind of function he was having that day in terms of, as far as I could tell, if I could tell, whether he was functioning normally that day, because, as I mentioned before, the recognition of the event is not the

same as the occurrence of the event.
Q. Is there any sort of limit to the temporality that you would require?
A. It would depend on the individual case, but I'm on record for saying that two, three days is not beyond the period of time where the effect of ephedra-related compounds can have a causative effect on brain hemorrhaging. In fact, I've said that it can be as much as two weeks before.
Q. All right. I want to ask you some questions about the effects, but before I do, let's finish going through sort of the background facts or history here. So if you still have that page of your report in front of you, page two, you say that the product label reads that each tablet contains 21 milligrams of concentrated ephedra extract and three milligrams of caffeine?
A. Yes.

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Shields
Q. The product contains, as you understand it, herbal ma huong, correct?
A. Yes.
Q. What do you mean by concentrated herbal extract?
A. I believe I quoted from some label or product information because that's not a term that I would ordinarily use.
Q. I think the term, I'll hand you back the label, Exhibit 10, uses the term "Ephedra group alkaloids, concentrated, 21 milligrams Ephedra group alkaloids and three milligrams concentrated caffeine in the form of herbal extracts."
Is that what you ear referring to?
A. Yes, I paraphrased that by saying 21 milligrams of concentrated ephedra extract.
Q. So you're referring to the 21 milligrams of ephedrine alkaloids referenced on the Herbalife label.
So is it your understanding then that that's contained in each tablet that he took and, that he took three tablets twice a day?
A. That's what I expressed as my

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Shields
understanding. So that would be all together 126 milligrams of ephedra extract.
Q. And do you believe there's a dose response relationship with respect to ephedra or ephedrine?
A. Well, let's put it this way. There's no dose that's without some potential for causing trouble. The higher doses probably are more troublesome, but there's many case reports where patients took relatively low doses of PPA.
The one that comes to my mind right off is the Hemorrhagic Stroke Project whereas doses as low as 7 milligrams of PPA were recorded as being related to intracerebral hemorrhage.
Q. I'm sorry. I didn't mean to cut you off.

A. Well, there's a lot more I can say about it, but ask me your next question.
Q. And I'm going to come back to the Hemorrhagic Stroke Project later, but the Hemorrhagic Stroke Project, you're referring to findings with respect to phenylpropanolamine, correct?
A. Correct, but there was a derivative

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study by Morganstern. He was the lead author which looked at ephedra products.
Q. Did that study make any findings at 7 milligrams of ephedra alkaloids?
A. First of all, that finding said, that study states that they didn't really have enough cases to make any kind of definitive statement, but 32 milligrams a day, there may be a relationship. That's basically what it says, and they did discover, which I think is the most important part of this, seven cases of intracerebral, intracranial bleeding, five subarachnoid, two intracerebral of people who had taken ephedra; and their conclusion was that above 32 milligrams a day -- I might have to take this.
Q. Okay.
(Discussion held off the record.)
THE WITNESS: So it was inconclusive, but the final idea there was, to me, the fact that that was important, is they did find those cases, and the conclusion was that you needed to have 32 milligrams a day or more. That was their conclusion,

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of ephedra-related as opposed to pure PPA.
MR. OETHEIMER:
Q. Now, they didn't actually, we'll come back to that, but they didn't actually find a statistically significant association, even above 32 milligrams?
A. Yes. Excuse me.
(Discussion held off the record.)
(Record read.)
THE WITNESS: Yes, because the study.
MR. OETHEIMER:
Q. That should be a yes, they did not?
A. The study was underpowered, number one, and, number two, both studies suffered tremendous flaws, and I'm not talking about the highly technological epidemiology comments because I'm not an epidemiologist. In the two groups, they excluded people who died and people who had serious neurologic injury, which is what happens, especially with sub arachnoid hemorrhage. So that's limitation, so that means that's also limitation on the interpretation, however they made it.
Q. Now, back to the dose, you referred

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to, let me just, this copy that you have,

unfortunately, as you said, parts of that are not legible, and you said you thought you might have other labelling information?
A. Yeah, I couldn't read this very well.
Q. This has, Exhibit 10 has a 2001 Herbalife copyright. I'm going to hand you a document.
MR. OETHEIMER: Why don't I mark this as Exhibit 11.
(Defendants' Exhibit 11, document, marked for identification, as of this date.
MR. OETHEIMER:
Q. I'll give you a moment to read it, Dr. Shields.
A. I might need a magnifying glass to read this.
Q. It seems to me it's a better copy than the one you were working on.
A. Well, I told you, I had a better copy.
(Discussion held off the record.)
THE WITNESS: Let's see now, the part

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Shields

that you're probably interested in, three tablets original green a day.
MR. OETHEIMER:
Q. Right, it's the portion that's not legible on Exhibit 10. It says, "Three thermogenic original green tablets contain 21 milligrams concentrated ephedrine group alkaloids and 3 milligrams caffeine in the form of herbal extracts."
Do you see that?
A. Yes.
Q. In your report, you indicated that you believed, at least at the time you wrote your report, that each tablet contained 21 milligrams of ephedrine alkaloids and 3 milligrams of caffeine.
That label indicates that's the total of three tablets?
A. That's one way of reading it.
Q. Do you have any basis for reading it differently?
A. I think that's the most reasonable reading of it. It's not stated very clearly.
Q. So that would make, if Mr. Singh was

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Shields

taking three green tablets twice a day, each serving, that would mean 21 milligrams of ephedrine alkaloids in the morning and another 21 milligrams in the afternoon for a total dose of 42, not 123?
A. But the dose could not change my opinion.
Q. Is there any dose below which you would change your opinion?
A. The issue is purely of dose. It would depend on what the other factors available were in an individual case.

Q. How about in this case?
A. In his case?
Q. Yes.
A. I would say that any dose, in his case, except 0, I would consider significant, given the events as related, a year of use and this dosage, yes.
Q. But as for that day, even 0?
A. Yes, yes. I made that totally clear.
Q. And just, on the left of that, the suggested serving, if you see that at the top left, "Take 1 to 3 original green tablets, one

00103

beige tablet twice a day at 10 a.m. and 4 p.m."
Do you know whether Mr. Singh followed that regimen?
A. Well, he was unclear, even in his deposition that I just read, as to what he was doing. Two or three times, I believe he said.
Q. But if he took three tablets twice a day and he followed this regimen so he'd taken the first three tablets at 10 a.m., the likelihood is that he would have already experienced the onset of his hemorrhage prior to 10 a.m., correct?
A. Possibly.
Q. Now, you reviewed Mr. Singh's deposition prior to issuing your December 4th, 2006 report, correct?
A. No, I said, yes, I had, before the December 4th, yes.
Q. And you went over, in preparation for the deposition, sometime within the past week to two weeks, you went over your file and you made some typographical corrections in your work?
A. I made handwritten corrections to typos.

00104

Q. Right, and you indicated that if you found anything that was in error in your report, you would obviously want to note that?
A. Yes.
Q. So at this point, can we agree that the statement, in terms of the dosage containing the tablet, the footnote, the asterisk on the second page of your report, it appears that may be in error, correct?
A. Yes.
Q. And the statement that Mr. Singh had taken Herbalife that morning, that's a statement that you cannot support at this time, correct?
A. I don't know if that's true. I wouldn't say I can't, I wouldn't use the term I can't support it. That was the history that I got.
Q. But you don't know if it's true?
A. Well, I wasn't there. You don't know if it's true or not either. You weren't there.
Q. But you're not basing your opinion on his having taken it that morning, are you?
A. Well, I believe I made that very

clear.

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Shields

Q. Can you make it clearer on the record?

A. Well, why ask the same question four times?

Q. Well --

A. I'll say it one more time because I don't want to be disagreeable, but I also don't want to sit here all day answering the same questions. It wouldn't make any difference to me in view of the facts here.

Q. You've been clear on that and I do understand that, and I'm going to ask your indulgence because my question is slightly different.

I understand it may not matter or effect your opinion, but what I want to just be clear on the record is that you are not basing your opinion at this time on it being established that he did take the product that day?

A. That's correct.

Q. We can turn to page three of your report, please.

A. Yes.

Q. You state that Mr. Singh had been a

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Shields

pack-a-day smoker for 30 years?

A. Yes.

Q. And I believe that was reflected in your interview notes?

A. Yes.

Q. One pack per day times 30 years, referring you back to Exhibit 6?

A. Yes.

Q. So that 30 years, we sort of relate that 30 years backwards from 2005 or from 2003?

A. I think it's back from 2005 because I believe there's another note saying that he quit six months before, so it would be --

Q. Six months, when you say six months ago in your November 2005 note?

A. It refers to six months ago --

Q. 2005?

A. Yes.

Q. So he continued to smoke after his stroke?

A. Yes.

Q. And you think the 30 years probably relates backwards from 2005, so we would have 28 years in 2003?

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Shields

A. Many years.

Q. Many years.

He's only 41 at the time of his stroke, so 28 years puts him back to age 13?

A. Yeah.

Q. And there's no dispute that that a history of smoking is a major risk factor for aneurysmal rupture and hemorrhagic stroke?

A. I believe I stated that in my report.
Q. What role do you believe it played in Mr. Singh's subarachnoid hemorrhage?
A. Well, there's two parts to that question; number one, long-term effect. It certainly was not good for him and certainly contributed to the weakness of the aneurism, the chronic smoking, but the most interesting part of all that to me is smoking on the day of the subarachnoid hemorrhage, that increases, the way I look at it is, the long-term increase in smoking risk for patients who have subarachnoid hemorrhage is actually two and a half to three times people who don't smoke who have aneurisms. If you smoke on the day, there's a nine times greater chance, and to me, that's highly

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significant.
According to the history that I got, he did not smoke on the day that he ruptured his aneurism, so that meant that the ongoing, two and a half to three and a half times risk was, in fact, a contributing factor, and it had that factor present for 20-odd years before he blew his aneurism. So it was there, so it made some contribution.
Q. Okay, the --
A. By itself, it didn't blow the aneurism.
Q. Is that conclusion based solely on the fact that his, that your history was that he did not smoke that morning?
A. No. If he had smoked that morning, then the effect of smoking -- I'm trying to be very clear.
Am I confusing you in some way?
Q. I don't believe so.
A. Well, I just really answered that. If he had smoked that morning, it would be the difference between a nine times greater risk and a two and a half to three and a half times

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greater risk. It would have been a more significant contribution.
Q. And I do understand that.
So it's a significant risk factor whether he smoked that day or not?
A. Absolutely, but it's much more if he did smoke that day.
Q. And now what I want to understand is your basis for believing he didn't smoke that day?
A. History.
Q. The history you took from either Mr. Singh or his wife when you interviewed him on November 14th, 2005?
A. Yes.
Q. You already told me that you don't consider his testimony about whether he took Herbalife that morning reliable because people who have a stroke are confused, Doctor.

So am I being unfair here in suggesting that it seems like you're picking and choosing and believing what you want?

A. I don't think you're being unfair in pointing it out that way, but I fully agree, it

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Shields

may not be accurate. I just want to point something else out.

I don't know if you're going to ask me about this, but since you're asking about smoking, here's my take on how that works. The effect, the really bad reason the smoking being responsible for the blowing of the aneurism on that day is its effect on a system which you might call the "elastase system."

Now, the elastase system basically destroys elastic tissue. If you smoke, you allow elastase to not be inhibited, and that's why it's bad for you on that day. It has other bad effects for you. It may increase your blood pressure systemically, et cetera; however, most of the effects of smoking actually go in the direction of producing clots rather than hemorrhages, but if you look at the half life of nicotine, which is about two hours, if he didn't smoke on that day, then you're talking about a ten-hour effect from his last cigarette for the elastase effect to be present.

Q. And is the elastase effect that transient that it's gone in hours?

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Shields

A. Well, it does what it does, and the next time you do it, you're exposed again.

Q. He had tea that morning?

A. Yes.

Q. Is it familiar to you as a human as well as a doctor that smokers are in the habit of having a cigarette with their tea or coffee?

A. No, I think the cigarette with coffee. I don't know about tea, could be.

Q. In any event, he had a cup of tea that morning, and your assumption is that it was caffeinated tea?

A. It is my assumption. I don't know.

Q. Assuming it was caffeinated tea, it may have had some effect on his blood pressure?

A. Not only blood pressure, but the effects of ephedra.

Q. Assuming he took it, which we don't know, assuming he took the ephedra that day?

A. Well, even if he took it the day before because the long-term effect of ephedra on blood vessels of the brain which I'm interested in is the vasoconstrictive effects and caffeine adds to vasoconstriction in the brain too.

00112

Shields

Q. How much caffeine is in a cup of tea?

A. Varies on how strong you make it, assuming it's caffeinated, 30 to over 100 milligrams, and if you make it superstrong,

it would be more than that.
Q. So those numbers sound comparable to coffee?
A. It's just like coffee.
Q. The effects of the blood vessel, let me start to go into this.
What is the mechanism by which, in your opinion is that the ephedra that Mr. Singh took, whether he took it that way or not, was contributory to his hemorrhage, but as I understand it, the smoking was also contributory?
A. Yes, just as I said in my report.
Q. What is the mechanism by which you believe ephedra played a role in contributing to the hemorrhage?
A. Ephedra produces cerebral vasospasm, and how does that happen, by alpha one constriction. Alpha one receptors are on the blood vessels of the brain, and when you stimulate them, they can contribute. Ephedra

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stimulates alpha one and beta one receptors on blood vessels, but the ephedrine effect is vasoconstriction, and if there's any PPA, you basically have, which these extracts inevitably contain some varying amounts, and some think that ephedrine is converted to PPA in the body, so PPA has a primarily vasoconstrictive effect. It is not damped down by the beta one effect or the beta two effects.
Q. Okay. Let me follow up. So I asked you what mechanism you believe is at work in the case.
A. Vasoconstriction.
Q. I'm going to come back and ask you about that.
So I didn't hear anything about increasing blood pressure?
A. Well, increasing blood pressure may be an effect, but I don't think that's the primary effect, especially if, in fact, he didn't take the medication that morning.
Q. So then let me ask you about cerebral vasospasm.
Is there any clinical evidence of that in

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the medical records?
A. Well, there is the following; first of all, you don't need clinical evidence of it.
MO MR. OETHEIMER: I move to strike.
MR. OETHEIMER:
Q. I didn't ask if you needed it. I'm asking if there's any evidence.
A. I'm explaining it to you.
MR. OETHEIMER: Okay, I move to strike the nonresponsive portions and ask you to continue.
THE WITNESS: And I will.
MR. OETHEIMER: Whether I like it or not.
THE WITNESS: Very often,

vasoconstriction will not be visible on imaging studies because the vasoconstriction may be so severe that the vessels become unangiographically recognizable or the blood vessels are too small to be seen in the first place. That's one thing.

Second, the vasoconstriction that's induced by these agents can also affect

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Shields

the veins, which are not typically looked at on these studies which drain and can have a back effect pressure on the vasculature. Thirdly, and I never noticed, and maybe it's present in the chart someplace, a repeat arteriogram which looked at what was called, just give me a second, fibromuscular dysplasia in the internal carotid artery because that could have been vasospasm.

So what I would have liked to have seen would have been a repeat study, and it may even have been done, I just didn't find it, which looked at what was present, actually, in the internal carotid artery in the next couple of days, because if it had been fibromuscular dysplasia, it should have been persistent for a period of time. So maybe that study is out there, I just don't know.

If the study doesn't show persistence of that deficit, which I'm not saying one way or the other, I'm just saying it might, I would consider it to be evidence

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Shields

of vasospasm. Secondly, there was evidence of vasospasm.

How do you think he infarcted the right side of his brain?

And To be completely fair, it may be that vasospasm happened because of the subarachnoid hemorrhage itself, which may be the original mechanism of vasospasm in some patients who have subarachnoid hemorrhages from vasospasm anyway who take ephedra because a small amount of blood expressed may be all that you see initially in a sub -- in an aneurism that bleeds.

That even rises to the level of a name, sentinal hemorrhage, which is recognized by the patient having headaches but obviously happens without the patient having headaches, so it could well be one of the mechanisms by which vasospasm is induced in the subarachnoid hemorrhage related vasospasm that's seen in patients who have aneurisms who take ephedra.

There are other reasons it can happen

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Shields

too. That's a very long answer. I'll summarize it. You don't have to see it. There was something that looked like vasospasm in the carotid on that side. There was never any follow up studies that showed that it was vasospasm. There was an assumption of FMD, fibromuscular dysplasia, and there was evidence the patient infarcted that right side of the brain.

MR. OETHEIMER:

Q. That was a long answer, and let me follow it up with a few, pin a couple of things down with that.

You don't dispute that on the report of the angiogram the finding was that there was no evidence of vasospasm seen?

A. I dispute that because there was an interpretation of an area that was called "fibromuscular dysplasia."

Q. Let me back up.

You don't dispute that the finding that was reported by the treating physicians was there was no evidence of vasospasm?

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Shields

A. You mean after he corrected himself? It was reported --

Q. You told me you have no reason to dispute that it was a typographical error.

A. No, I don't dispute it. I'm answering your question.

Q. I know you're getting worked up here, but --

A. I'm not getting worked up.

Q. The doctors in the medical records did not report finding evidence of vasospasm, correct?

A. No, I just told you. In the medical records, it says, "vasospasm."

Q. Where does it say vasospasm?

A. I showed it to you before in the note that I wrote.

Q. The one that was corrected?

A. Yes.

Q. So you accept now with the correction that the treating doctors, Dr. Zablow, and you read his transcript, that he found no evidence of vasospasm?

A. That's what he said.

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Shields

Q. Correct?

A. I'll agree that's what he said, but I don't agree that's what he found.

Q. You've already told me, and I want to follow up and ask you about each of the things that you just testified to.

So first, in terms of the finding and what he identified as fibromuscular dysplasia, or FMD, you've said, and I think I understand your answer, you said that could be evidence of vasospasm?

A. Might be.
Q. But you don't know?
A. No.
Q. You would have liked to have seen a test that wasn't done, as far as you know?
A. That's right.
Q. So you can't base an opinion on a finding that wasn't made, correct?
MR. RHEINGOLD: Objection.
THE WITNESS: It's a little too complicated for me to answer.
MR. OETHEIMER:
Q. Well, your opinion, you're not

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telling me that your opinion depends for its validity on that being vasospasm rather than fibromuscular dysplasia?
A. No, that was an answer to a question that you asked.
Q. I understand, and I'm following up on that question.
A. Here's my opinion. I don't know if it was vasospasm that was recognized in the neck. I don't know if it was vasospasm, FMD or some other condition. Nevertheless, it doesn't change my concept.
Q. Okay. Now, onto the next one, the right side. You said there is evidence of vasospasm in the records.
There was vasospasm on the right side of the brain subsequent to the hemorrhage, right?
A. Well, wait a minute. There was evidence of, the question you asked me was there evidence of vasospasm.
Q. I understand.
A. Now, as I recall, I'll look at my note, but the infarct on the right side of the brain was seen on the CT scan, not on an

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angiogram, so the inference of the vasospasm was, and it's on the right side of the brain, distant really from where the aneurism blew, That that was due to vasospasm. That's an inference. It isn't demonstrated, but it's evidence.
Am I making myself clear?
Q. Yes.
A. Because that's not the question you asked me.
Q. I understand, and the question I'm asking you now, which I think you already told me --
A. I want to make sure. I'm pretty sure that it was seen on a CT scan.
(Witness perusing document.)
It was seen on a CT scan.
Q. You're referring to the CT scan of May 13th, correct, Doctor?
A. The one that's clearest appears to be on May 24th.
Q. And it is commonly understood that vasospasm is a common or at least not atypical

response or sequelae to subarachnoid hemorrhage --

Shields

MR. RHEINGOLD: Objection.

MR. OETHEIMER:

Q. Occurring within 4 to 20 days afterwards, I think I've seen that.

Do you agree with that?

THE WITNESS: Should I answer despite your objection?

MR. OETHEIMER:

Q. He hasn't instructed you not to answer it. You should answer it.

A. I'd be happy to answer it. Yes, they typically occur, the vasospasm typically occurs no sooner than four days after the initial ictus, however, four days, there are variations. Four days is a little bit early, but it could be.

Q. You referred me to a CAT scan on May 24th, so that's two weeks after the event?

A. Yeah, but the one on the 13th also.

Q. Does the one on the 13th report the vasospasm?

A. None of them report vaso -- that's what I'm trying to tell you.

Q. All right.

A. There's hemorrhage in the right basal

Shields

ganglia and right thalamus. This doesn't specifically refer to the effect of vasospasm.

Q. I think I understand, and I just want to confirm my understanding of what you've already told me, which is, you pointed out that you're doing this, you have a CAT scan, not an angiogram.

So while it may be suggestive of vasospasm, it's not a finding of vasospasm, per se, correct?

A. Yes.

Q. So far so good?

A. Yeah.

Q. Secondly, because this is after the event, even if it is vasospasm, it may have only occurred subsequent to the subarachnoid hemorrhage and response to it, correct?

A. Yes.

Q. So if I go back and I ask the question, and maybe I should have been more precise in my original question and asked you whether there is evidence in the medical records of vasospasm existing at the time of the initial hemorrhage on May 10th, 2003, I take it you would

Shields

direct me to the finding of the internal carotid artery in the neck which was qualified Fibromuscular dysplasia, which you can't say?

A. You can't say it's FMD.

Q. And you haven't reviewed the films?

A. You can't say from those films whether it's FMD.

Q. How can you say if you haven't seen the films?
A. Because all it shows is as an irregularity in the blood vessels. There are lots of other reasons for it, and somebody that has taken a drug that can induce or a compound that can induce vasospasm, that's in the differential diagnosis.
Q. Now that may be one more thing before we leave the subject.
That finding, what Dr. Zablow referred to as fibromuscular dysplasia, was not in the intracerebral circulation?
A. It wasn't demonstrated there.
Q. Where was the finding that he noted?
A. I believe in the left carotid artery, in the neck, which is are where it's usually

00125

Shields

seen.
Q. What is usually seen?
A. FMD. I want to be complete, when it's seen in the cerebral circulation.
Q. Right.
So, again, to refine my question a little further, if I ask you if there is any evidence you can point to in the medical records of vasospasm existing at the time of the initial hemorrhage on May 10th, 2003, in the intracranial circulation, is there anything you can point me to?
A. There's no angiographic finding that supports that, which doesn't mean that it's excluded.
Q. What findings would support that, your view, as I understand it, it's the blood vessel, yeah, what would we, your opinion or your view is that ephedra can have an effect on the blood vessels?
A. It's not my view. That's well known.
Q. Is it your view?
A. Of course, but I don't want you to make it appear that it's just my view.

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Shields

You know, that's a little disingenuous, don't you think.
Q. You're testifying to your opinions, and that's all you can testify to?
A. But I can testify to what's generally understood, that which is what I'm doing.
Q. What findings, in other words --
A. What radiographic findings would there be?
Q. Right.
A. Well, one thing would be very thin wire-like vessels where the vessels were of a smaller caliber than they should be. Another would be a beaded pattern.
Another would be a very thin pattern of flow beyond the area of vasospasm, and the last thing would be the presence of collateral circulation, or you may see nothing whatsoever,

but a corrugated appearance, as is described in the internal carotid artery in this case, is also consistent with vasospasm.
Q. I'm sorry, the last, could you read that back to me?
A. Corrugated appearance.

Q. What blood vessels?
A. As was described in the internal carotid artery in this case.
Q. The internal carotid artery in the neck?
A. In the neck. You seem to be getting confused.
Q. No, I'm not confused. I just want the record to be clear.
This is what Dr. Zablow called the fibromuscular dysplasia?
A. Yes.
Q. And I understand you may see nothing whatsoever, but I just want to understand, is this a case where, other than that finding of the corrugated appearance in the carotid artery in the neck, is this a case where we see nothing whatsoever in the brain itself, or is there any other finding you can point to, whether a beaded pattern or any of the angiographic findings you just pointed to?
A. None are reported. I haven't seen the films myself.
Q. The beading pattern, is that

something that you would expect a competent neurologist to note in a report?
A. If it were present.
Q. And the fact that it was not noted then would suggest to you that it was not present?
A. Probably not.
Q. What results in the beading pattern?
Maybe you can just explain what you mean by the beading pattern.
A. Like a string of beads, areas of abnormal widening and areas of abnormal dilatation. Dilatation and widening are the same thing. Constriction.
Q. One quick thing, I refer, this is the top of page three of your report, we talked about whether that cup of tea that morning, you say, "Usually imbibed a cup of tea and two alcohol drinks per day"?
A. Yes.
Q. Again, I believe his testimony was that he consumed more like three cups of tea a day. Let me see if I can find that for you.
I'll show this to you, you had asked me if

I knew of errors to call to your attention, so I direct you to pages 234 and 235 of Mr. Singh's deposition transcript.

(Witness perusing document.)
A. So it says one to three.
Q. One to three, and he also says that he drank Lipton tea?
A. Yeah.
Q. And presumably, it doesn't indicate that it was caffeinated. You gave a range before of caffeine content of teas and said it can vary as to how it's brewed.
Any range on Lipton tea?
A. It depends on how you brew it.
Q. Steep it longer, it's going to be stronger.
Any indication that Mr. Singh, having an Indian background, that he drank it stronger?
A. Not to my knowledge, could be.
Q. There's also in your report a reference to his consumption of alcohol?
A. Yes.
Q. And I think, what did you say, two drinks per day?

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Shields

A. That's what he said.
Q. Is that a risk factor for hemorrhagic stroke?
A. It slightly increases the likelihood of hemorrhagic stroke, but the thing that really with alcohol, it really increases the likelihood of hemorrhagic stroke is binge drinking, heavy drinking, so I consider that negligible in this case, the alcohol effect.
Q. That presents some increased risk of hemorrhage?
A. It theoretically does, but at this level, it's a negligible effect. It's binge drinking that you really --
Q. I was going to say, you rely on his testimony was two drinks a day, beer, wine.
I'm sure in your own practice, you are familiar with the underreporting syndrome when it comes to alcohol consumption?
A. Yeah.
Q. At what point would you, is anything short of binge drinking, would anything short of binge drinking be a significant contributor?
MR. RHEINGOLD: Objection to form.

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Shields

THE WITNESS: Well, you have to look at the way in which alcohol promotes intracerebral hemorrhage. It does partially because people who drink alcohol often smoke. So if you start to winnow out the things that it does, it produces bouts of hypertension and more importantly interferes with liver function; and you need a good liver to make the coagulation factors that effect you.
So if you have low alcohol, which in this case it appears to be, then it becomes relatively unimportant unless you binge drink.

MR. OETHEIMER:
Q. Do you know whether Mr. Singh ate on the morning of his stroke?
A. I don't know.
Q. Is that important for you to know?
A. Well, anything, any piece of information could be important. Offhand, I don't see any important aspect to it that springs to mind, but if you have information, I'm happy to incorporate it.

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Q. I'll represent, I believe his testimony was that he had not eaten breakfast that morning.
A. I don't think that makes much difference.
MR. OETHEIMER: Why don't we take five minutes off the record.
(Discussion held off the record.)
(Time noted: 3:02 p.m.)
(Recess taken.)
(Time noted: 3:09 p.m.)
MR. OETHEIMER:
Q. Doctor, as we've discussed, Mr. Singh's subarachnoid hemorrhage resulted directly from rupture of an aneurism, correct?
A. Yes.
Q. And is that, in fact, how most subarachnoid hemorrhages occur?
A. About 85 percent. 75 to 85 percent happen because of an aneurysm.
Q. And do you know whether that aneurysm was congenital or developmental?
A. Well, congenital and developmental, the way I use the terms are the same thing.

00133

Q. Okay. I'm using the term "congenital" as maybe not medically appropriate, something that existed from birth?
A. Well, these are all considered to be congenital saccular aneurisms, which are developmental. So the defect, in most cases, is present from birth, but it's with the development of the vascular tree and the stresses that are applied to it that the aneurism becomes important. That's why the frequency of rupture increases with increasing age, even though the peak age is 55 or 50, 50 to 60, and it's rare to see an aneurism that ruptures before the age of 20.
It's rare to find aneurisms before the age of 20, but they're very plentiful in the population. So developmental and congenital are the same thing. Whereas, if you use the word "developmental" in the sense of acquired like from a mycotic aneurism, then you can say they're developmental, but that's not how the term is used.
Q. Do you agree with this statement: "Subarachnoid hemorrhage accounts for only five

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Shields

percent of strokes but occurs at a fairly young age"?

A. First of all, I don't agree to anything that I don't read myself. Secondly, that's completely opposite to what I just said because it doesn't define what a young age is, and I told you what the peak age of rupture is, so if you consider that to be young, to me, that's not young, that's middle aged.

Q. Let me ask you actually one follow up question that I meant to ask you before. In your long range answer, you made reference to sentinal headache or sentinal hemorrhage.

Do you recall that testimony?

A. Yes.

Q. Is there any clinical evidence that in this case Mr. Singh suffered from a sentinal headache prior to the morning of May 10th?

A. There's no clinical evidence of it.

Q. Is there some other evidence?

A. That's all I know is clinical evidence. I'm a clinical neurologist.

Q. If you tell me there's no clinical evidence, there's no evidence?

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Shields

A. That doesn't mean that. There may be other things to know about. For example, if this patient had been operated on, I mean, the old fashioned way, and you found old blood lying around the aneurism, that would mean there had been a sentinal hemorrhage. I don't have that information.

Q. Because they put coils in here. They didn't operate to clip the aneurism.

A. Right. They did electrocoagulation.

Q. You have no evidence that there was a sentinal headache the morning of May 10th?

A. That's what I just said. It couldn't be more clear.

Q. I'll show you this article and we can mark it.

(Defendants' Exhibit 12, document, marked for identification, as of this date.)

I don't think I have, I just have one copy with me. This is a review article published just several weeks ago in the "Lancet" entitled "Subarachnoid Hemorrhage."

We'll have to share one copy. I'm not going to take you through it, but I do, since I will

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Shields

ask you, in terms of incidence, I'll ask you whether you agree with a couple of the statements that appear here.

MR. RHEINGOLD: Just for the record, since we don't have copies, can you cite the full name, the author?

MR. OETHEIMER: It's titled "Subarachnoid Hemorrhage," the British spelling, from the Lancet Volume 369, dated January 27th, 2007, the actual, so

it's Lancet 2007, Volume 369, pages 306 to 318.

THE WITNESS: February 27, 2007?

MR. OETHEIMER: Did I say February?

January 27th, 2007.

MR. OETHEIMER:

Q. I think it said aneurisms are the cause of subarachnoid hemorrhages in 85 percent of cases.

Do you agree with that, not as a percentage, but as a ball park?

A. I believe I said that.

Q. Although it says that, "Although the incidence increases with age, half the patients

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Shields

are younger than 55 years at the time of subarachnoid hemorrhage."

Do you agree with that?

A. Well, that's a statistic you can discuss. It depends on what series you look at.

Q. I've seen it referred before that age 55 is sort of the mean.

Would you agree that many subarachnoid hemorrhages occur in people below age 50?

A. Some do.

Q. In your report at page, the top of page ten --

A. Ten.

Q. Actually, the sentence I want to ask you about begins the bottom of page nine.

A. Yeah.

Q. The statement is that, "However, the natural history of most cerebral aneurisms is overwhelmingly to never rupture; out of the approximately 15,000,000 cerebral aneurisms harbored in the U.S. population, there are only about 30,000 ruptures per year."

Do you see that?

A. Yes.

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Shields

Q. That would be the risk of population walking around with aneurisms, how many are going to have a rupture in a given year?

A. Yes.

Q. The lifetime risk would be greater; those people are walking around the aneurisms year after year?

A. That's true, but one other thing, since we're being very precise. First of all, there are those who think that aneurisms, this is basically on the assumption that aneurisms occur in approximately 6 or 7 percent of the population. There are studies which suggest that it might be, especially if you consider small ones, as many as 17 percent of the population have aneurisms, that's number one.

Number two, amongst the 30,000, I just rounded that out, most estimates are 28,000, but there could be 30,000, I wouldn't doubt that. A certain percentage of that is reruptures. So the number is even probably, in terms of probability,

greater.
Q. Let me ask, many people have very small aneurisms?

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Shields
A. That's correct.
Q. Never --
A. Well, theoretically, aneurisms start out small and they get bigger, and that's what has to do with all these factors in life, including those of just --
Q. Including smoking?
A. Including smoking and taking drugs and compounds like ephedra. We're talking about hemodynamic and wearing down effects. That's why increasing age is a risk for blowing your aneurism.
It's a wearing down process, but anyway I'm going to your question of the size. They're all small at one time, most of them. Almost all of them were small at one time.
Q. As they grow, does the risk of rupture increase?
A. Yes.
Q. All things being equal?
A. That's well known.
Q. What other factors would dispose an aneurism to, you know, increase the likelihood of rupture?

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Shields
A. Well, I started to go into that before. Binge drinking; cigarette smoking, especially on the day; use of drugs like ephedra-containing products; alterations of blood pressure; chronic blood pressure and acute blood pressure elevations; wearing down processes; jetting of flow; alteration of direction of flow; pulsatile flow; turbulence of flow; vibration of the aneurism; pregnancy; sex; disease; activity; lack of activity. I can go on for a very long time.
Q. You have, and I apologize. My question was imprecise. We did go over all that.
A. I didn't get to tell you all that.
Q. Actually, in my last question, I was focussing, and I apologize, it was not clear. I meant to focus on whether there were things about the aneurism itself, in terms of its size, location, appearance, that predispose to rupture from whatever instigating factors may result in that.
A. So what is your question?
Q. Other than size, you've told me that increased size increases the likelihood of

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Shields
rupture, right?
A. Yes.
Q. Are there other things about the aneurism, itself, in terms of where it is in the brain, in terms of the aneurism itself that would make it more likely to rupture?

A. Well, the most important factors in terms of its rupturing are physiologic and dynamic factors, as I just started to reel off. I take it that you mean, is there some structural aspect of the aneurism which would make you think it would tend to rupture.

Q. Or are aneurisms in a particular area of the brain more likely to rupture than another?

A. Well, aneurisms in the subarachnoid space are more likely to rupture than other aneurisms, and aneurisms that have obviously thin walls are prone to rupture; and aneurisms that have previously ruptured tend to rupture, and the only physical characteristic of an aneurism that's been shown in studies that tells you whether an aneurism is going to rupture or not is, in fact, the size.

And teats don't matter, and if you like,

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I'll give you the references to save ourselves a lot of trouble. So you might want to write this down; Thorolf Sundt, T-H-O-R-O-L-F S-U-N-D-T, New England Journal of Medicine, I don't remember the issue, but it's easy to find. And also Lokesely, L-O-K-E-S-E-L-Y, in "The Cooperative Study Of Aneurysms And Arteriovenous Malformations," and it's also in some commonly read textbooks. I believe Lou Kaplan's.

Q. Stroke?

A. I think it's in his, but anyway, the classical references I gave to you.

Q. First of all, in terms of size, the size of Mr. Singh's aneurism?

A. It's in the range of the most common size that ruptures.

Q. 7 to 10 millimeters?

A. Yes, five to 10 are the most commons size, although, if you have a bigger one, you have a bigger chance, but there are more 5 to 10ers around than giant aneurisms.

Q. How about the location. I've got a, this is a review article from the New England Journal of Medicine, New England Journal of

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Medicine, Volume Five, pages 928 to 939?

A. Who is the author?

Q. I'll hand you a copy.

A. By Schipp?

Q. Review Article On Cerebral Aneurisms.

A. Yes.

Q. Do you know the author?

A. Yes.

Q. Really, I found this useful, and also I'd like to direct you to the second page, page 929, since we don't have the films here in front of us, perhaps you can use this Figure One to locate where you understand Mr. Singh's aneurism?

A. Well, let's see, they say it's a lobular -- they have an exact description here.

MR. OETHEIMER: I'll mark this article as Exhibit 13.

(Defendants' Exhibit 13, document, marked for identification, as of this date.

THE WITNESS: Well, according to this, this is at the, seven by five, for the left internal bifurcation aneurism. That would be right here.

Shields

MR. OETHEIMER:

Q. Right, and that would be pointing to?

A. I'll make a circle if you want. That was the description. As I said, I wasn't there.

Q. And you've circled the internal carotid artery bifurcation?

A. Yes.

Q. And that's where the internal carotid artery basically splits, and the middle cerebral artery goes in one direction and the interior cerebral artery goes in another?

A. Yes.

Q. That's one of the areas that aneurisms commonly form?

A. Well, they all, the greatest number of them are in that area. I don't mean that area, the anterior part of the circle of willets is where they commonly form.

Q. And do they commonly form at points of bifurcation?

A. Yes.

Q. What percentage of those form at areas of bifurcation?

A. A high percentage. I don't know the

Shields

exact percentage.

Q. Are aneurisms at a point of bifurcation at greater risk of rupture than aneurisms that are not?

A. You'd have to be more specific. The peripheral aneurisms tend to rupture not as often.

Q. Now, the references that you gave me, as I understand, are on the issue of teats, whether --

A. On what physical elements of the aneurism is predictive of blowing.

Q. And --

A. There are others too. Those are just the most classic.

Q. What physical elements are predictive of rupture?

A. Size.

Q. How about you mentioned thinness?

A. Thinness of the dome.

Q. Of the dome. What I understood from Dr. Zablow's testimony, and you obviously, you can tell me what you think, but what I understood him to say is that this particular aneurism of

Shields

Mr. Singh had two teats or domes, correct?

A. Not domes, the teats are projections

from the domes.
Q. Is the teats?
A. From the dome, singular.
Q. Okay, one dome, two teats?
A. In this case. Well, I don't want to be petty, but this was a lobular, so there was more than one dome.
Q. What does lobular mean?
A. It's like, lobular, it's like a three leaf clover.
(Discussion held off the record.)
MR. OETHEIMER:
Q. The teats project from the dome?
A. Yes.
Q. Are the teats area where the vessel or where the surface is stretched even thinner?
A. Yes.
Q. So are those areas that are predisposed or disposed to rupture?
A. Well, those are the areas which might rupture, and they may be on the way to rupture, but you can have teats without them rupturing,

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Shields

and the studies that I mentioned before show that teats, per se, do not predict rupture; and a lot of thinking of this is derived from the old way of trying to figure out which aneurism bled, but we don't have to do that anymore. And also, well, anyway that's all I have to say about that.
Q. So, teats, per se, your view, may not --
A. Predict rupture.
Q. Predict rupture?
A. But the thinness does. That is an area where the rupture might occur, but they don't predict that they're going to rupture.
Q. Do you recall --
A. And also, if you have a condition which is challenging the blood vessel wall, like an alteration of flow, turbulent flow, jet flow, vibratory flow, pulsatile flow, it may push out a teat; and that may be a weakening of the wall, but that's an effect of the wearing down process, whatever that wearing down process is that's producing that wearing down. So that would be quite consistent with the use of ephedra over the course of the year, so that doesn't change

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Shields

anything.
Q. Do you know --
A. And if you think that the teats were always there, you have the situation of smoking for 28 years with teats and never a rupture, so it's illogical.
Q. Do they get increasingly thin with wear and tear?
A. They might. It depends on what happened. Actually, there's ways in which the smoking protects the teats because smoking produces clotting, and if you get some clotting at the orifice of the teat, it may, in fact,

protect the teat.
Q. Do you recall Dr. Zablow's testimony regarding his physical observations concerning these teats?
A. I'd have to read it.
Q. And I'll hand it to you, for the record. He described a finding that he had two, I call it "teats." Teats, he has teats or domes.
"Question: Are they also just intimal lining?
Answer: They're intimal lining that's

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Shields

already become thinner, so they're extremely thin."
That's page 64 of Dr. Zablow's deposition. I would also direct you to page 67.
A. Well, first of all, he didn't visualize that. You mean he was able to look inside the teat?
He was talking about what the arteriogram looked like. The presumption is the teat is an area of thinning.
Q. You're saying he could not have visualized it?
A. That's right. He could visualize the presence of the teat and infer from that that is a point of weakness, which is obvious.
Q. Based on that, I don't need to show this to you, but I'm happy to read it to you.
A. I'd rather see it. I like to see it for myself.
Where is it?
Q. Page 64, I read from, between pages 64 and 67. I believe he testifies regarding that subject.
(Witness perusing document.)

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Shields

A. I see what he says. Here's one of the things. This may save some trouble.
One of the things that I saw Dr. Zablow said is not in agreement with what everybody else thinks, which is, on page 66, he says, "In the United States, per year there are about 60,000 subarachnoid hemorrhages of which 50,000 are probably ruptured aneurisms," and this thing that you cited yourself, oh, no, you didn't cite it. He's 100 percent wrong.
Q. Well, you think his numbers are overstated but his percentages are right?
A. The percentage of subarachnoid hemorrhages --
Q. Which are due to ruptured aneurisms?
A. Which is in the eighties. I told you before, it's a level of information. He also says three percent. Seems adequate to me, but it's okay. He says the frequency is three percent, which most people, as I say, will think it's 5 to 9 percent and some as high as 17 percent. I accept the five percent.
Q. While we're on Dr. Zablow, let me ask you this.

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Shields

Having reviewed the medical records, do you have any criticism of the treatment Mr. Singh received?

A. I would say no, but there are some things that are missing that I would like to know about.

Q. What are those?

You've identified --

A. The repeat angio, if there were one, and there were a lot of blood tests that I would have liked to have seen that I didn't get, lab studies, but on the whole, it seemed to me he was very well taken care of.

Q. The one lab study, let me actually ask you a question about that because I skipped over that.

On page three of your report, Exhibit 1, there was a tox screen done, correct?

A. Yes.

Q. And it was negative for everything reported there?

A. Yes, it was negative for amphetamines, which is the one we'd be interested in, in this situation, but the amphetamine

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Shields

determination for ephedra products depends on the method that's used, and you, I don't even know, the most modern way to do this, but the old way to do it was if you were really looking for ephedra, you would have to do gas chromatography, which may have been done here. I doubt it. It's a very old fashioned way to do it. So the negatively of amphetamine doesn't tell you anything.

Q. Without knowing what the lab protocol was, you really don't know whether this tells you anything.

In other words, we know it's negative, nothing's reported?

A. No, you're overstating it. There are lab procedures which, when you're testing for amphetamines, may show the presence of ephedra, but they don't necessarily show that. So negative doesn't mean as much as positive.

Do you understand?

Q. Mm-hmm.

A. This doesn't tell me whether there was ephedra there or not. If it were positive, I might say there was ephedra there because they

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Shields

might do a secondary test.

Q. Your point is having simply a reporting that is negative for amphetamine on this tox screen does not rule out the presence of ephedra in his bloodstream. It doesn't establish the presence, but it doesn't rule it out.

A. Doesn't do anything.

Q. However, if we knew more about how that tox screen was done, we might know the

answer to that question?
A. Most of the modern tox screens which test for amphetamines do not show the ephedra.
Q. But you have not done any investigation to determine how this one was done?
A. I don't know.
Q. A few sort of the stray odds and ends, let me ask you, blood pressure, are there diurnal variations in blood pressure?
A. Yes.
Q. And, typically, when does systemic blood pressure peak in most people?
A. It varies. It's highly dependent upon, in fact, on the economic status. Your blood pressure tends to universally fade during

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the night and then comes up during the day. Then it peaks at various times, depending upon your attitude towards your life.
For example, strokes occur more commonly on Mondays in people who have low socioeconomic status. That's because they don't like their jobs and they're anxious about it. That's related to their blood pressure. So I can't generalize beyond that.
Q. Is it fair to say that the morning, say between the hours of 8 and 10 a.m. in the morning at least typically tends to be a period of higher blood pressure readings in most individuals?
A. Yes, in general, in general.
Q. Blood pressure fluctuates?
A. From instant to instant.
Q. So in terms of transient increase in blood pressure, we all have them, all the time?
A. Yeah, you're having them now.
Q. One of your prior answers, you mentioned that the stress -- I may not have this exactly, so I'm happy to have you sort of clarify, but the stresses and forces, you made

00155

reference to jetting in the internal cerebral vasculature, correct?
A. Internal cerebral vasculature, cerebral vasculature.
Q. Yes.
A. Yes.
Q. Do you believe that played a role in Mr. Singh's hemorrhage?
A. Yes.
Q. Do you believe that ephedra had some effect on the vessels in his brain?
A. Yes.
Q. And how does that, the wear and tear that you refer to, the wear and tear of the blood flow through the vessels, then, how does that impact that?
A. Well, if you have an area of weakness in your system, like an aneurism, and I don't only confine it to aneurisms, but in the instant case, aneurisms, if you have an agent which

produces alteration in the caliber of blood flow around that structure and does it even intermittently, it will produce an alteration of the radiologic nature of the blood flow. It will

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Shields

cause areas of turbulence, it will cause vibration. It will cause areas of direction of blood flow, in fact, that's a good way to get a teat. It's like focussing a fire hose on a point.

Turbulence, vibration and adds to pulsatile flow. It produces areas of faster flow and slower flow and also areas of increased pressure, and that's obvious. It's like dropping a rock in a stream. On top of that, the area around an aneurism typically has a defect in auto regulation, and you need autoregulation in order to protect vasculature in the brain; and the effect of ephedra producing vasoconstriction tends to override the autoregulation which makes the weakened part even more vulnerable to excesses flow, all those things I just said and all those hemodynamic effects.

Q. Does it make any difference, Dr. Zablow, in his testimony, and I will show this to you on page 36, "The one thing that is evident from the angiogram is the problem is not a hemodynamic flow related problem involving the brain in the sense that the cerebral circulation

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is put together in a way that one of the carotid arteries is the dominant to the two carotid arteries and is supplying a disproportionate amount of vascular territory in the brain, then there may be hemodynamic consequence in regards to the arteries that are in the neck that come off the aorta to supply the brain. In this particular circumstance, the dominant carotid artery circulation is actually contralateral right side, so that would indicate then the changes in the carotid artery on the left side would not the result of hemodynamic stress on the carotid artery so rather there's." He goes on.

Do you agree or disagree that where this aneurism was located was not the area where basically the blood was dominantly flowing, and does that have any bearing on your opinion?

A. I think what he says is utter nonsense, and I totally understand what his point is, but is the, your understanding that he's saying that this vessel, this vessel weakness, the aneurism, was not a result of blood flow; then why would it ever blow if it doesn't have a hemodynamic basis to it?

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Shields

I think he kind of lost himself in there.

Q. Do you, let me just ask, do you agree --

A. Wait a minute. Let me read this again. I'm trying to make some sense of it.

Maybe it's transcribed wrong.
Well, the question was, "Did the appearance of the artery tell you or suggest to you what period of time that flow problem had been in existence?"
"Answer: No, the one thing that is evident from the angiogram is that the problem is not a hemodynamic flow problem in the sense that the cerebral circulation is put together in a way that one of the carotid arteries is dominant."
I don't agree with it. Given its most sentient possibilities, I don't agree with it.
Q. Do you agree that the area where this aneurism was found is not where the dominant carotid artery circulation to the brain is?
A. I'd have to see the arteriogram myself to make that determination.
Q. You used the term, and we'll clean up, in the report you used the term "segmental

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constriction."
A. Yes.
Q. That's what we've been talking about?
A. Vasoconstriction.
Q. Then I don't need to ask you anything further?
A. I explained why it's segmental because the muscular wrapping around the blood vessels is not continuous, it's more like a spiral, so that when you get a constriction, you get areas of narrowing and areas of dilatation. You don't get a flattening unless it's extremely severe.
Q. You make reference, while looking here on page eight, you make reference to an amphetamine.
Amphetamine and ephedrine and phenylpropanolamine are all, as I understand, that are all sympathomimetic compounds?
A. Yes.
Q. You would agree, however, that there are significant differences between various sympathomimetic compounds?
A. I do. For example, alpha one and

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beta one constriction is greater with ephedrine than it is with amphetamines. Amphetamines is a more exciting, cerebral excitatory agent than any of the others, but it doesn't have the same vasoconstrictive effects, although it does have a greater capability of producing arteritis, And I'll give you a reference for that too. That's in Gilman and Gilman.
Q. But would you agree you cannot take studies involving amphetamines and unthinkingly translate them to phenylpropanolamine or ephedrine?
A. It's reasonable to analogize, we do that all the time in medicine. We're not saying that's exactly the same, but you have a model that works. So it's reasonable to accord some of

the same characteristics to a compound. I gave you a reference to that too in the bibliography, the extra one that I gave you. It's reasonable to analogize from one drug to another.

Q. Exhibit 4?

A. Might be the next one, maybe not. If it isn't here, I'll give it to you. Well, you

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can look at both of these over here, but there's another, there's another better reference for it. Anyway, the reference only supports my notion of this, which is that it is reasonable to analogize similar compounds in terms of their effects, and I'll give you that reference if you want me to.

Q. Okay.

A. But I think you'll find it in the Lasagna articles too, Louis Lasagna.

Q. You say cigarette smoking was a predisposing risk factor?

A. Yes.

Q. Just explain to me what you --

A. I think it weakened the wall of the aneurism, and I think it also probably made this man subject to surges of blood pressure, like any smoking would do, which is part of the wearing down effect.

Q. When you say surges of blood pressure, any increase?

A. Systemic blood pressure.

Q. Such as could occur with a cup of tea?

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A. Yes.

Q. Or exercise?

A. Well, exercise is complicated. Exercise, typically, will increase systolic blood pressure, not so much diastolic. In some people it reduces blood pressure.

Q. Okay?

A. But that's a complicated thing.

Q. I'm not sure how much he exercised. How about sexual relations?

A. Well, exercise and sexual relations do have an effect, but just as many blown aneurisms occur in sleep and just sitting around.

Q. And the normal diurnal variations in blood pressure?

A. Yeah, but it's impressive, I mean, it's impressive what happens when somebody's lifting weights, where we have the Valsalva effect and we see dramatic alterations in blood pressure.

Q. You referred earlier to the Morganstern paper, and I do have a copy of it.

(Defendants' Exhibit 14, document, marked for identification, as of this

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date.

MR. OETHEIMER:
Q. There you go, Exhibit 14.
(Witness perusing document.)
A. What about it?
Q. I just wanted to give you a chance to look at it. This is, frankly, Doctor, this is more for the record, since we referred to it earlier; you referred to it in your testimony, and I asked you a question or two about it, but this is the Morganstern paper which you referred to earlier?
A. Yes.
Q. Which was an outgrowth of the Hemorrhagic Stroke Project, the paper that was published where they looked at the data they had with respect to ephedra and hemorrhagic stroke?
A. Yes.
Q. And the specific finding, at least in the abstract here, was ephedra was not associated, at least they did not find a statistically significant association between ephedra and hemorrhagic stroke, although they noted a trend at doses above 32 milligrams.

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Shields
Is that what you referred to earlier?
A. Yes. Well, if you read it, it's exactly what it says. This is the abstract.
Q. Well, if we're going to read it exactly, I guess, do you know if the authors wrote the abstract or the New England Journal did.
A. I don't know.
Q. Why don't we refer to the paper. I know refer to the abstract.
A. The abstract is typically written by the authors and might be edited by the editors, that's the way it's usually done, but exactly what happened here, I don't know.
Q. In the discussion --
A. But to read the abstract, the last sentence says, "Ephedra is not associated with increased risk for hemorrhagic stroke except possibly at higher doses," and then the paper itself, I believe, it says, "For daily doses less than, equal or less than 32 milligrams a day, the relative risk was 1.54, was, let's see, was .13, but it was 3.59 for doses above 32 milligrams a day." That's what the abstract says. Now, I

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Shields
don't think the report says anything different that than.
Q. Right. The report says in the discussion section, it says, "Although the overall results did not indicate an association between the use of ephedra-containing products and increased risk for hemorrhagic stroke, the analysis by dose suggests there may be an association with use of more than 32 milligrams daily."
See that?
I was on page 134.

A. Yeah, I am on. I don't see it.
Q. The first sentence --
A. Oh, yeah.
Q. Under discussion.
A. I'm looking here. Well, it also says, "Bias is an unlikely explanation for our finding in association between doses of ephedra greater than 32 milligrams a day and risk for hemorrhagic stroke."
Q. Right. They did not find a statistically significant association?
A. They explained why. They didn't have

00166

enough people.
Q. They didn't have enough people. There may be reasons, but they didn't have data to make a finding.
A. You don't want to over interpret.
Q. Right.
Do you rely at all on the main Hemorrhagic Stroke Project paper on phenylpropanolamine in the presence of hemorrhagic stroke?
A. Only that it demonstrated that people who took PPA get hemorrhagic strokes and that a high percentage are subarachnoid hemorrhages.
Q. Do you know what the average dose was?
A. I don't remember the average dose, but the range was 6 to 150 milligrams.
Q. I'm going to hand you this paper, "Phenylpropanolamine And Risk Of Hemorrhagic Stroke."
MR. OETHEIMER: Mark that as the next exhibit, 15.
(Defendants' Exhibit 15, document, marked for identification, as of this date.)

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MR. OETHEIMER:
Q. Dr. Shields, I know you've seen the paper before, and you're happy to review as much of the paper as you like. I'm going to ask you about a paragraph that appears on page 1829.
A. I'm on 1829.
Q. In the right-hand column halfway down, there's a paragraph that begins, "We also examined the possibility of a dose effect."
A. Yes.
Q. Why don't you read that paragraph to yourself and then we can talk about it?
A. Okay.
Q. So it reflects here that the median dose of PPA in the study was 75 milligrams.
Do you see that?
A. That's correct.
Q. And whatever their findings, their findings were the odds ratio was higher for doses above the median dose of 75 milligrams?
A. Yes.
Q. Do you --
A. Well, it isn't clear to me here

whether we're talking about 75 milligrams a day,

which is what I think it is, but go ahead and ask the question.

Q. I'm not sure we need to belabor it. My only frame of reference here was seeking to understand that the average dose or mean median dose in the PPA study was significantly above, you know, a daily, recognizing that we're talking about phenylpropanolamine on one hand and ephedrine on the other, but just in terms of doses, we've got 75 milligrams versus Mr. Singh's daily dose of perhaps 42 milligrams of ephedrine alkaloids.

It's roughly double?

A. So what's the question?

Q. Do you agree that this looked at higher doses, that the Hemorrhagic Stroke Project --

A. It does say that, but we're looking at this selectively, and as I told you before, the doses ranged from 6 to 150 milligrams, and also the study, as I told you before, is terrifically flawed by the fact that it excluded people who died and people who had serious neurologic impairment. So it definitely says

what you just said, 75 milligrams.

Q. Whatever the flaw is, way back this morning you made reference to This Hemorrhagic Stroke Project and that strokes, I think you indicated that strokes had occurred or were noted in people that had taken as low as seven milligrams or something of phenylpropanolamine?

A. Six.

Q. Six, but there were no statistical findings.

A. These statistical findings, even these are valid.

What's the point?

(Discussion held off the record.)

(Defendants' Exhibit 16, document, marked for identification, as of this date.)

MR. OETHEIMER:

Q. Doctor, one more paper from the Hemorrhagic Stroke Project, not one that I'm, I think the other two you had cited, perhaps, in your bibliography. I'm not sure if this one appears.

A. It probably does. I'm familiar with

it.

Q. You're familiar with it in any event? This is the review study.

A. Oh, now I see where he got the 55 to 60,000 patients.

Q. You think this is where Dr. --

A. Well, it says, "Subarachnoid hemorrhage and intracerebral hemorrhage affect

55,000 to 60,000 people a year."
Q. You're referring to where Dr. Zablow got his numbers, and you don't know where he got his numbers?
A. Probably similar.
Q. In the Hemorrhagic Stroke Project, they were only looking at hemorrhagic strokes?
A. Yes.
Q. They were looking at subarachnoid hemorrhages and intercerebral hemorrhages?
A. Yes.
Q. There are similarities between the two and differences between the two?
A. Between what?
Q. Between intracerebral hemorrhages and subarachnoid hemorrhages.

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Shields

A. Right.
Q. If I could direct you to page six, just quickly for the numbers, the final case group for the HSP comprised 702 subjects, including 425 translating to 60 percent with subarachnoid hemorrhage, the other 40 percent with intracerebral. And of the 425 cases of subarachnoid hemorrhage, 312 met the criteria for aneurysmal subarachnoid hemorrhage," correct?
A. Yes.
Q. Based on this paper, that's more like 75 percent than 85 percent, but that's still the range?
A. It's a range.
Q. And this paper simply reports, based on the comparison of those 400, actually 312 cases of aneurysmal subarachnoid hemorrhage and matched controls findings regarding the relative risk for major risk factors, correct?
A. Yes.
Q. And the authors state, I believe, you've got the highlighted version, I should make this the exhibit.
Why don't you substitute that?

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Shields

MR. OETHEIMER: We'll let you mark it in a moment.
MR. OETHEIMER:
Q. The authors state on page 1377 that "The odds ratio for the association with risk for aneurysmal, SAH, subarachnoid hemorrhage, was highest for family history of hemorrhagic stroke and current cigarette smoking."
Do you see that?
A. No.
Where are you reading?
Q. I'm reading from the paragraph on the right-hand column at the bottom of the page below the table on page 1377. Begins, "In the multivariable model."
A. Yes.
Q. Other than a family history of hemorrhagic stroke, the highest risk factor reported for aneurysmal subarachnoid hemorrhage

is cigarette smoking, correct?
A. Yes.
Q. And you don't disagree with that?
A. Well, I think hypertension is a more significant factor, but I already said that I

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consider it significant.
Q. And there's nothing to indicate in this paper that the significance of smoking as a risk factor for aneurysmal subarachnoid hemorrhage depends on whether the subject smoked on the day of the stroke?
A. Yes, but there are a lot of other studies which do show that.
Q. And have you cited those?
A. Not specifically, I don't think so.
Q. Can you direct me to those references?
A. Sure. Just write it down.
Q. In any event, the relative risks reported here for smoking of 3.7, well, odds ratio of 3.73 is consistent, I think, with what you testified earlier?
A. I said 2.5 to 3.5.
Q. Correct, in the absence of smoking on the day of the stroke and nine or something?
A. Well, the nine would probably bring it up to the 3.73.
Q. On the left-hand up on that page, it says, "With regard to health behaviors and use of

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medicine, cases were more likely to be current cigarette smokers and heavy alcohol," and it looks like it says, "Equal to or greater than two drinks daily"?
A. Yes.
Q. "And caffeine, greater than two caffeine drinks daily"?
A. Yes, but you have to parse out the alcohol, like I said before in terms of clotting factors and effects on blood pressure and the concomitance of the smoking.
Q. And I think I understand that, but just in terms of an analysis, controlling, they found in this study that drinking alcohol at greater than or equal to two drinks daily, itself, was an independent risk factor with an odds ratio of almost 3, correct, looking at the table?
A. Let's see. Alcohol, I don't see the odds ratio.
Q. Matched?
A. Matched odds ratio, I see it. Yes, I see that. I don't agree with it, though.
Q. And caffeinated drinks at greater

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than or equal to five a day is a matched odds ratio of just below two?
A. Yes.
Q. Lower than alcohol, but higher than

phenylpropanolamine, which is reported at an odds ratio of only 1.15.
A. I don't believe that this particular paper discusses people who use both.
Q. People who use both what, I'm sorry?
A. Caffeine and ephedra products, PPA, in this case.
Q. One of the flaws that you found in this study, the HSP, was that they did not include fatalities, correct?
A. Yes.
Q. If I could direct you to page 1380, and actually, you may want to read the carryover paragraph. It begins at the bottom of 1379, "Subsequent Control Case Study."
A. Where are we?
Q. Page 1379, the bottom right-hand column.
A. 1379.
Q. It's the paragraph that begins --

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A. Yes, I see that.
Q. If you would, you can read that paragraph to yourself.
A. Yes, I've seen this.
Q. So the authors are saying there --
A. There was a study where they looked at the patients who died.
Q. They're saying that the distribution of risk factors is documented in the medical record of those fatalities from subarachnoid hemorrhages was similar to what they came up with here?
A. Yeah, but the study didn't have enough numbers. Yes, I'm aware of that.
Q. Have we covered all of the opinions you would expect to give in this case?
A. As far as I can think of.
Q. Have you been asked to form opinions regarding Mr. Singh's damages?
A. I don't think I've been formally asked, but I've given opinions on it.
Q. And to the extent in terms of his residual deficits?
A. Yes, it's in my report.

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Q. I was going to say, it's in your report and we can say and I can take you through that, but if you're willing to stipulate that any opinions you have or any information you have about his current condition, or at least his condition as of November 2005 is contained in your report?
A. I certainly can stipulate to that.
Q. You examined him on November 14th, 2005.
Do you have any current information on how Mr. Singh is doing?
A. In your report, you note that his physician, Dr. Hirschfeld, at some point several months after the stroke had pronounced him

essentially fit to return to work, but that when you interviewed Mr. Singh, that you did not feel capable of working. I did not think that he could work.

Q. That was my question.
Did you form, do you have a view?
A. Well, let me see what he said.
Q. It's at page four of your December 4th?

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Shields

A. Yeah, this is what I said on page 14. "He is permanently to severely disabled in the fullest sense. He is unable to work at his usual profession and cannot enjoy the fruits of life."
Q. What is it that impairs his ability to work at his usual profession?
A. Multiple, impairment of mental function, impairment, as I remember, he did fine work with silver, and I found that both his hands were extremely clumsy.
Q. He repaired jewelry, and you found that his fine motor skills were impaired?
A. Yes.
MR. OETHEIMER: All right. I have no further questions at this time. There are a number of references that Dr. Shields has agreed to supply. Some of them he may recall and some of them he may need to be prompted.
THE WITNESS: I need a note.
MR. OETHEIMER: I will send a note to Mr. Rheingold.
MR. RHEINGOLD: Yes, please do that.
MR. OETHEIMER: And I am going to

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Shields

keep the deposition open only for that purpose, since those references are not available to me today. It's not my expectation that it will be necessary to call you back to ask you questions about those, but I'm going to reserve my right to do so.
MR. RHEINGOLD: When the weather gets better.
MR. OETHEIMER: Thank you very much.
THE WITNESS: Thank you.
(Time noted: 4:14 p.m.)

_____
LAWRENCE SHIELDS, M.D.

Subscribed and sworn to before me
this ___ day of _____ 2007.

_____

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C E R T I F I C A T E

STATE OF NEW YORK)
) Ss.
COUNTY OF SUFFOLK)

I, JEAN VALERIE GAFA, a Notary Public within and for the State of New York, do hereby certify:

That LAWRENCE SHIELDS, M.D., the witness whose deposition is hereinbefore set forth, was duly sworn by me and that such deposition is a true record of the testimony given by the witness.

I further certify that I am not related to any of the parties to this action by blood or marriage, and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 23rd day of February, 2007.

_____
JEAN VALERIE GAFA

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I N D E X

EXHIBITS

	Exhibit 11	1-page photocopy of Herbalife label	100
	Exhibit 12	13-page article entitled "Subarachnoid haemorrhage"	135
	Exhibit 13	12-page article entitled "Cerebral Aneurisms	143
	Exhibit 14	4-page paper entitled "Use of Ephedra-containing Products and Risk for Hemorrhagic Stroke	162
	Exhibit 15	7-page article entitled "Phenylpropanlomine and the Risk of Hemorrhagic Stroke	166
	Exhibit 16	8-page article entitled "Major Risk Factors for Aneurysmal Subarachnoid Hemorrhage in the Young are Modifiable	169

ERRATA SHEET

NAME OF CAPTION: SINGH VS. HERBALIFE
DATE OF DEPOSITION: FEBRUARY 20, 2007
NAME OF WITNESS: LAWRENCE SHIELDS, M.D.

PAGE	LINES	FROM	TO

_____
LAWRENCE SHIELDS, M.D.

Subscribed and sworn to before me
This ____ day of _____ 2007.

_____
(Notary Public) My Commission Expires